REMARKS/ARGUMENTS

The Status of the Claims.

Claims 1-13 and claim 16 are pending with entry of this amendment. Claims 1 and 16 are amended herein to more completely identify antecedence of a term within the claims. These technical amendments do not add subject matter and do not change the scope of the claims. Claims 14 and 15 have previously been cancelled and claim 16 has previously been added.

35 U.S.C. §112, Second Paragraph.

Claims 1-13 and 16 are rejected under 35 U.S.C. §112, second paragraph, for alleged indefiniteness based on the Examiner's assertion that "it is not clear when a cognate receptor is not an estrogen receptor."

The Examiner asks a question (Office Action, page 3) outlining the theory on which the rejection is based: "[I]f the cognate receptor is one species of estrogen receptor[,] then is an estrogen receptor[,] which is a fusion protein or truncated protein[,] different from the cognate receptor which is an estrogen receptor"[?] The question does not need to be answered because it is irrelevant to the present claims, and cannot support the present rejection. As a preliminary matter, the word "different" does not exist in the claims, so is irrelevant to the discussion. Moreover, the premise of the question - "if the cognate receptor is one species of estrogen receptor", is literally and specifically excluded as a possibility in the claims. Claim 1 is limited to methods comprising providing a cell comprising "a cognate receptor for the nuclear transcription factor ligand, which cognate receptor is not an estrogen receptor", emphasis added. Because the cognate receptor is not an estrogen receptor, by definition, it cannot be "one species of estrogen receptor." Although the phrases of the Examiner's question are ambiguous, the terms of the present claims are not. Claim 1 is clear and distinct concerning the fact that the cognate receptor is not an estrogen receptor.

A cognate receptor that is not an estrogen receptor is distinct from an estrogen receptor. The difference is well understood by those skilled in the art and as discussed in the present specification. Estrogen receptors are members of a large family of nuclear receptors

that have many similarities in peptide sequences and functional domains. However, one skilled in the art understands that the distinguishing characteristic between different receptors is the ligands they bind. American Heritage Dictionary - Receptor: "Biochemistry. A molecular structure or site on the surface or interior of a cell that binds with substances such as hormones, antigens, drugs, or neurotransmitters." (Emphasis added.) In the case of fusion proteins or truncated proteins, for example, they may be considered estrogen receptors, or not, depending on whether they bind estrogen. This basic understanding of the defining relationship between receptors and ligands is clear in the art and apparent throughout the present specification. The proper starting place in any claim construction analysis is the language of the claims themselves, read in view of the specification and the prosecution history (see, *Phillips v. AHW*). In particular, estrogen receptors, including truncated forms and chimeras, are discussed at paragraph 64. At least one thing the cited list of estrogen receptors have in common is that they all bind the ligand estrogen. According to the state of the art and the present specification, a cognate receptor that does not bind estrogen, would not be an estrogen receptor. The metes and bounds of the terms could not be more clear and simple. The section 112 rejection should be withdrawn.

Furthermore, although the Examiner has not argued directly against the presence of the negative limitation in the claims, Applicants reiterate that the proviso wherein "the cognate receptor is not an estrogen receptor" is entirely proper and well supported by the specification according to *In re Johnson*, 558 F.2d 1008, 1019, 194 USPQ 187, 196 (CCPA 1977).

Claim 16 was also rejected under section 112, second paragraph. However, the Examiner never discusses the basis for the rejection and it should be withdrawn. Applicant notes that claim 16 does not include the term "which cognate receptor is not an estrogen receptor".

Because the claims are definite and particularly point out and distinctly claim the subject matter which Applicants regard as the invention, the Applicants respectfully request the section 112 rejections be withdrawn.

35 U.S.C. §102.

Claims 1-5 and 8-11 were rejected under 35 U.S.C. §102(e) as allegedly anticipated by Kushner, et al., 5,723,291. Claims 1-13 and 16 were rejected under 35 U.S.C. §102(e) as allegedly anticipated by Evans, et al., 5,639,592. Claims 1-2, 4, 8, 10-11 and 16 were rejected under 35 U.S.C. §102(e) as allegedly anticipated by Pfahl, et al., 6,004,748. Applicants traverse.

In order for a reference to anticipate an invention, the reference must teach each and every element of the claimed invention. That is, anticipation requires that "all limitations of the claim are found in the reference, or 'fully met' by it." Kalman v. Kimberly-Clark Corp., 218 USPQ 781, 789 (Fed. Cir. 1983). With respect to independent claim 1, and the rejected dependent claims, all limitations are not found in any of the cited references.

Not all limitations of claims 1 and 16 are found in any of Kushner, Evans or Pfahl. For each of the three references, the Examiner rejected independent claims 1 and 16 based on the erroneous contention that the "limitation of cognate receptor is generic and encompasses additional modified estrogen receptors or fos and jun proteins in the cell." However, Review of the present claims, in view of the specification, the understanding of one skilled in the art, and the plain meaning of the terms, reveals a variety of limitations not disclosed in any of the cited references. For example, see claim 1:

> 1. A method of screening a nuclear transcription factor ligand for an ability to modulate estrogen activation at an AP-1 site, said method comprising the steps of:
>
> a) providing a first cell, **in the absence of said nuclear transcription factor ligand**, comprising:
>
> a cognate receptor for the nuclear transcription factor ligand, **which cognate receptor is not an estrogen receptor**;
>
> an estrogen receptor;
>
> fos;
>
> jun; and,
>
> a promoter comprising an AP-1 site that regulates expression of a first reporter gene;

b) contacting said first cell with said transcription factor ligand and with a compound having AP-1 mediated estrogenic activity; and,

c) detecting expression of said first reporter gene, as compared to expression of said first reporter gene in the absence of said transcription factor ligand, wherein a difference in expression of said first reporter gene in the presence and absence of said transcription factor ligand indicates that said nuclear transcription factor ligand modulates estrogen activation at an AP-1 site.

Each of the cited references essentially includes a cell containing a hormone receptor (such as an estrogen receptor) that can interact with AP-1 proteins (such as fos and jun) to induce transcription of a reporter gene promoted by an AP-1 site. None of the references discuss a cognate receptor for a transcription factor ligand that is not specifically listed as being comprised in the cell of the claim. None of the references discuss contacting the cell with the transcription factor ligand which is to a cognate receptor that is not an estrogen receptor. None of the references discuss comparing expression of the reporter gene in the presence of the transcription factor ligand to expression of the reporter gene in the absence of the transcription factor ligand. The Examiner fails to allege, for each cited reference, the presence of several components and method step limitations required in the claims, thus failing to state a *prima facie* case. Because these limitations are not present in the cited references, claim 1, claim 16, and the dependent claims cannot be anticipated by the references. Therefore, the rejections should be withdrawn.

As a preliminary matter, the present allegations and section 102 rejections are essentially identical to those of the previous Office Action. The rejections were traversed with support of substantial written counter arguments in the previous Response of August 5, 2005. Yet, the prior response was almost entirely ignored in the present Office Action, but for the general cursory pronouncement that all prior arguments were "unpersuasive". Applicant directs the Examiner's attention to MPEP 707.07(f) Answer All Material Traversed, wherein an examiner must provide clear explanations of all actions taken by the examiner during prosecution of an application ... the Examiner is instructed, "if he or she repeats the rejection, [to] take note of the applicant's argument and answer the substance of it." The section 102 rejections are repeated but no answer has been provided specifically to

the substance of Applicants' position. In the interest of making progress in this application, Applicants request the answer to this Response be in compliance with MPEP 707.

Again, the Examiner alleges the "limitation of cognate receptor is generic and encompasses additional estrogen receptors or fos and jun proteins in the cell". The Examiner alleges that "both fos and jun are nuclear receptors and ligands because they are transcription factors and they bind each other." These interpretations are unreasonable in light of the specification as a whole, the plain meaning of the terms in the art and arguments of the previous Response. Applicants again note that claim 1, requires that the cognate receptor that binds the transcription factor ligand is not an estrogen receptor. According to the Examiner's logic, if either fos or jun were considered a cognate receptor, the other would have to be considered the associated transcription factor ligand. However, the cell of claim 1, clause a), specifically comprises both fos and jun in the absence of the associated transcription factor ligand. Therefore, neither fos or jun can be the transcription factor ligand. Therefore, neither fos nor jun can be the cognate receptor of the claim. Because the erroneous identification of fos or jun as cognate receptors is essential to the section 102 rejections, the rejections should be withdrawn.

On page 4 of the Office Action, the rejection is supported by the allegation essentially stating that estrogen receptor fusion proteins of Kushner '291 can be a "cognate receptor" that "is not an estrogen receptor". However, this flies in the face of the legitimate negative limitation in the claim and the clear meaning of estrogen receptors, as discussed above with regard to the section 112 rejections. The Kushner '291 estrogen receptor fusion proteins bound estrogen and were thus, by definition, estrogen receptors. Therefore, the cited Kushner '291 estrogen receptor fusion proteins are indeed estrogen receptors and are excluded from the cognate receptors of the claim. Because the Kushner '291 estrogen receptors, or any other estrogen receptors, specifically excluded as "cognate receptors" of the claim, the section 102 rejections must be withdrawn. With regard to dependent claims, Kushner '291 does not describe: the second cell with an ERE and the cognate receptor of claim 2; a cell with an ERE and the cognate receptor of claim 3; the second cell of claim 4; the cell of claim 5, e.g., with a cognate receptor and a second reporter; a factor ligand to a cognate receptor of claim 6; a cognate receptor of claim 7; a cognate receptor expressed from

a heterologous DNA of claim 9; a progestin cognate receptor of claim 12; or, a glucocorticoid cognate receptor of claim 13.

Evans '592 describes a cell containing a hormone receptor that controls expression of a reporter through AP-1 proteins at an AP-1 site: essentially the classic AP-1 mediated reporter system common to several cited references. In the case where the hormone receptor is an estrogen receptor, Evans describes essentially the assay system of Kushner '291. Evans also describes cells containing certain cognate receptors that are not estrogen receptors, such as a glucocorticoid receptor. However, Evans does not describe at least the limitations of providing a cell that in the absence of a nuclear transcription factor ligand to a cognate receptor includes fos, jun, and an estrogen receptor; contacting the cell with a compound having AP-1 mediated estrogen is activity (estrogen is not mentioned in Evans); contacting the cell with a transcription factor ligand to the cognate receptor that is not an estrogen receptor (Evans' test compounds all appear to be transfected receptor proteins, i.e., transcription factors, not transcription factor ligands); or, comparing reporter expression in the presence and absence of the transcription factor ligand. The list of Evans disclosures, on page 5 of the Office Action, does not completely describe the claimed invention and thus does not meet claim 1 limitations. Rejections for alleged anticipation by Evans should be withdrawn. Moreover, Evans fails to disclose at least the additional limitations of several dependent claims, such as, e.g., the second cell with an ERE and the cognate receptor of claim 2; a cell with an ERE and the cognate receptor of claim 3; the second cell of claim 4; the cell of claim 5, e.g., with a cognate receptor and a second reporter; a factor ligand to a cognate receptor of claim 6; or, a progestin cognate receptor of claim 12.

Pfahl describes "a method of inhibiting the transcription of a gene, which is activated by AP-1 or an AP-1 component, comprising binding AP-1 or the component with a nuclear receptor so as to prevent the binding of AP-1 to the gene." See abstract. The Examiner suggests that because Pfahl used dexamethasone to interact with glucocorticoid receptors, the present invention is allegedly described. However, dexamethasone activates the glucocorticoid receptor, which inhibits the signal from the phorbol activated reporter system. In the Examiner's example, a reporter gene controlled by AP-1 proteins is activated chemically (i.e., by activation of the protein kinase C pathway with a phorbol ester), and not

by an estrogen receptor; therefore, Evans does not disclose modulation of estrogen activation of and AP-1 site, as required by the claims. From Applicant's review of the Pfahl system, it fails at least to disclose: a method for screening for nuclear transcription ligands; a method in which estrogen activation of an AP-1 site is modulated by a ligand to a cognate receptor which is not an estrogen receptor; contacting a cell with both a ligand to the cognate receptor and a compound having estrogen mediated estrogenic activity; and detecting expression of the reporter gene to determine any modulation of the ligand on estrogen activation of the AP-1 site. The Examiner must specifically identify where these limitations are allegedly present in Pfahl, or must withdraw the rejections based on Pfahl. With regard to dependent claims, Pfahl does not describe: the second cell with an ERE and the cognate receptor of claim 2; a cell with an ERE and the cognate receptor of claim 3; the second cell of claim 4; the cell of with a cognate receptor and a second reporter of claim 5; an estrogen receptor expressed from a heterologous DNA of claim 8; a cell expressing fos or jun from a heterologous DNA of claim 10; or, a progestin cognate receptor of claim 12.

In a side note to the section 102 rejection, the Examiner alleges that claims 2 and 3 do not further limit claim 1. However, Applicants would like further clarification of this allegation because the claims do appear to provide at least the further limitations of: 1) a second cell, 2) a promoter comprising an ERE, and 3) a second reporter gene.

Because several required limitations are missing from the cited references, Applicants request the rejections for anticipation be withdrawn for claim 1, claim 16, and all dependent claims.

Additional limitations of dependent claims are not found in the cited references. The Examiner has apparently rejected claims 2 and 3 based on the statement that they do not further limit rejected claim 1. However, this is not the case for the claims, as stated above, which include limitations not found in claim 1 and not described in the cited references. For example, with regard to claim 2, the cited references do not provide at least the limitation of a second cell comprising the cognate receptor for the nuclear transcription factor (not necessarily comprised in the first cell), or the limitation of contacting the second cell with the nuclear transcription factor ligand, or the limitation of detecting expression of a second reporter gene.

With regard to claim 3, the cited references fail to provide the limitation wherein the first and second cell are the same cell.

Claims 4 and 5 were apparently rejected again because they "do not further limit claim than the receptor in claims 2 and 3." Applicant again does not understand the basis for this statement. Applicant notes, however, that claims 4 and 5 include limitations not required by claim 1 and not found in the cited art. Claims 4 and 5 also contain different limitations from claims 2 and 3, and have a scope different from claims 2 and 3. For example, the additional limitations of claims 4 and 5 over claim 1 include at least a second cell comprising a cognate receptor of the transcription factor ligand (which is not necessarily comprised in the first cell), a response element that regulates a second reporter gene, contacting the second cell with the transcription factor ligand, and detecting the second reporter gene. With regard to claim 5, the cited references additionally fail to provide the limitation wherein the first and second cell are the same cell. This contrasts with claim 2, which provides a second cell comprising the cognate receptor of claim 1 (which is not an estrogen receptor) and an estrogen response element, these limitations are not found in claim 4.

Because many limitations of independent claim 1 and dependent claims are not present in the cited references, Applicants respectfully request the rejections for anticipation be withdrawn.

35 U.S.C. §103(a).

Claims 1-13 were rejected under 35 U.S.C. §103(a) as allegedly obvious based on Kushner in light of Pfahl, Evans, Gaub, Webb, and Kushner (WO95/06754). Applicants traverse.

Three requirements must be met for a *prima facie* case of obviousness. First, the prior art reference must teach all of the limitations of the claims. M.P.E.P § 2143.03. Second, there must be a motivation to modify the reference or combine the teachings to produce the claimed invention. M.P.E.P. § 2143.01. Third, a reasonable expectation of success is required. M.P.E.P. § 2143.02. The teaching or suggestion to combine and the expectation of success must be both found in the prior art and not based on Applicants' disclosure. M.P.E.P. §2143.

Specifically, a *prima facie* case of obviousness requires that the combination of the cited art, taken with the general knowledge in the field, must provide all of the elements of the claimed invention. When a rejection depends on a combination of prior art references, there must be some teaching, suggestion or motivation to combine the references. In re Geiger, 815 USPQ2s 1276, 1278 (Fed. Cir. 1987). Moreover, to support an obviousness rejection the cited references must additionally provide a reasonable expectation of success. In re Vaeck, 20 USPQ2d 1438 (Fed. Cir. 1991), citing In re Dow Chemical Co., 5 USPQ2d 1529, 1531 (Fed. Cir. 1988).

No combination of cited references provide all the limitations of any claim rejected for alleged obviousness. The cited references are cumulative and discuss essentially the same basic technology of a cell containing a hormone receptor (such as an estrogen receptor) that can interact with AP-1 proteins (such as fos and jun) to induce transcription of a reporter gene promoted by an AP-1 site. The discussion of missing limitations required to state a *prima facie* case of obviousness is essentially the same as the missing limitations arguments made above with regard to rejections based on alleged anticipation (see above). None of the references discuss the additional limitation of a cell containing an estrogen receptor and a cognate receptor for a transcription factor ligand that is not necessarily present in the cell in the presence of the listed elements (e.g., fos and jun). None of the references discuss contacting the cell with both the transcription factor ligand and the compound having AP-1 mediated estrogenic activity. None of the references discuss comparing expression in the cell of the reporter gene in the presence and absence of the transcription factor ligand. Because these limitations are not present in any combination of the cited references, claim 1 claim 16, and the dependent claims can not be obvious in light of any combination of the cited references.

None of the estrogen receptors, fos, or jun, can be the "cognate receptor" of the claims for the transcription factor ligand, as discussed above. Neither fos or jun can be the transcription factor ligand, as discussed above. Therefore, the cited references fail to provide a cell with both an estrogen receptor and a cognate receptor, as required by independent claim 1.

Again, as described above, claims 2, 3, 4, and 5, are further limiting over claim 1 and include limitations not found in the cited references. For example, Gaub, not cited as anticipating claim 1, fails for provide limitations required by the claims and not present in other cited references. Gaub, as cited on page 1271, provides cells comprising an estrogen receptor and estradiol inducing transcription of a reporter gene through fos and jun at an estrogen responsive element (ERE), e.g., in the presence of a phorbol ester. Gaub fails to provide many of the same limitations not found in Kushner, Evans or Pfahl, as discussed above. Gaub fails to provide a method of screening a nuclear transcription factor ligand to a cognate receptor (which is not an estrogen receptor) for an ability to modulate estrogen activation at an AP-1 site. Gaub further fails to describe at least, e.g., a cognate receptor for a transcription factor ligand that is not listed as comprised in the cell and which is not an estrogen receptor; contacting the cell with the transcription factor ligand to the cognate receptor; comparing expression of the reporter gene in the presence and absence of the transcription factor ligand; a second cell comprising the cognate receptor for the nuclear transcription factor (absent from the first cell); contacting the second cell with the nuclear transcription factor ligand; detecting expression of a second reporter gene; or expression of two different reporter genes in the same individual cell. The final Webb and Kushner references are admittedly cumulative and provide no additional limitations. The rejections of all claims based on alleged obviousness should be withdrawn for failure of the combined references to describe all the limitations of the claims.

The cited combinations of references are not motivated, and thus are not obvious according to established case law. Obviousness can only be established by combining or modifying the teachings of the prior art to produce the claimed invention where there is some teaching, suggestion, or motivation to do so found either explicitly or implicitly in the references themselves or in the knowledge generally available to one of ordinary skill in the art. See, *In re Fine*, 837 F.2d 1071, 5 USPQ2d 1596 (Fed. Cir. 1988). Here, there is not a suggestion in any of the references to make a combination that provides the claimed inventions. In fact, no express or implicit teaching or motivation can exist to combine references forming an invention when the references themselves do not cumulatively include all the necessary limitations of the invention.

Evans is cited, at columns 1 and 2, as motivating the present inventions. However, Evans directs one to the study of interactions between various receptor proteins and not the screening of ligands, or the present invention. This misdirection is only accentuated by the further cited alleged motivation of Gaub, which again, is focused on receptor protein interactions and interactions with nucleic acids, but not screening or interactions with ligands. Pfahl suggests his methods can be useful in screening for new ligands, at the top of column 2. However, such screening methods are never described. Pfahl teaches away from the present invention by directing the poisoning of the system and making the present methods non-functional, e.g., by application of phorbol esters. Again, there can be no motivation in a combination that does not provide all the necessary limitations.

If the proposed modification or combination of the prior art would change the principle of operation of the prior art invention being modified, then the teachings of the references are not sufficient to render the claims prima facie obvious. In re Ratti, 270 F.2d 810, 123 USPQ 349 (CCPA 1959). Assuming *arguendo* that the references could provide all the limitations of any rejected claim, the suggested modification to the primary reference (Kushner) would necessarily change the principle of operation substantially. The suggested changes to the estrogen detector of Kushner (patent '291) would require a substantial reconstruction and redesign of the assay system and a change in the basic principle under which the Kushner system was designed to operate. Kushner operates, e.g., by activating AP-1 proteins to promote translation of a reporter at an AP-1 site in the presence of estrogen. With theoretical modifications to provide claim 1, the system would have to change in operation, e.g., from a direct signal output assay to a new and different modulated signal output resulting from new interactions with additional (e.g., cognate) receptor systems. The changed system would operate to results unrelated to the assay results of the original Kushner '291 technique.

There would be no expectation of success in the cited combinations. Combinations of cited art would not be expected to succeed in providing the presently claimed inventions because they are missing critical limitations for the function of the screening methods. Furthermore, the presence of chemicals, such as phorbol esters found in many of the cited

references, would create reporter signal noise rendering modulation by cognate receptors undiscernable or incapable of interpretation in the screening methods.

With reference to the side argument presented on page 8 of the Office Action, claims 4 and 5 further limit claim 1 and are not of the same scope as claims 2 and 3, as discussed above. Therefore, claims 4 and 5 should be examined with particularity.

Obviousness type Double Patenting

Claims 1-13 and 16 were also rejected under the judicially created doctrine of obviousness-type double patenting over claims 1-27 of the '291 patent in light of Pfahl, Evans, Gaub, Webb and Kushner as indicated above. An obviousness type double patenting rejection is appropriate if the claimed invention, while not identical, is not patentably distinct with respect to the claims of a prior patent in light of the prior art. A claimed invention is not patentably distinct if all of the claimed elements are found in one or more pieces of prior art, and if there is motivation to combine the prior art with a reasonable expectation of success. Because the claims are not obvious, as discussed above, they can not be subject to this double patenting rejection.

Claims 1-13 and 16 of the present invention relate to a method of screening a nuclear transcription factor ligand for the ability to modulate estrogen activation at an AP-1 site. The elements of the method are:

a) providing a first cell, in the absence of said nuclear transcription factor ligand, comprising:

a cognate receptor for the nuclear transcription factor ligand, which cognate receptor is not an estrogen receptor;

an estrogen receptor;

fos;

jun; and,

a promoter comprising an AP-1 site that regulates expression of a first reporter gene;

b) contacting said first cell with said transcription factor ligand and with a compound having AP-1 mediated estrogenic activity; and,

c) detecting expression of said first reporter gene, as compared to expression of said first reporter gene in the absence of said transcription factor ligand, wherein a difference in expression of said first reporter gene in the presence and absence of said transcription factor ligand indicates that said nuclear transcription factor ligand modulates estrogen activation at an AP-1 site.

The '291 patent relates to a different method for screening a different type of test compound involving providing a cell comprising: AP-1 proteins (e.g., fos and/or jun); an estrogen receptor; and, a construct comprising an AP1 site which regulates expression of a reporter gene. None of the claims of the '291 patent recites, e.g., a cell with both an estrogen receptor and a cognate receptor to an transcription factor ligand that is not fos or jun, or contacting the cell with both the transcription factor ligand to the cognate receptor and a compound having AP-1 mediated estrogenic activity, or comparing expression of a reporter gene in the presence and absence of transcription factor ligand, as is found in independent claim 1 of the present invention. Additional limitations not found in the specification of the '291 patent, as discussed above, are also not present in the '291 claims.

The Examiner has not pointed to anything in the cited references that provides all the limitations missing from the rejected claims. Applicants have not found these limitations with reasonable efforts. The cited references cumulatively provide, e.g., individual hormone receptors that activate expression of a reporter gene through an AP-1 protein/receptor element pathway. As discussed above in the arguments against the obviousness rejections, no combination of the same cited references with Kushner '291 provides any of several limitations including, e.g., a method of screening a nuclear transcription factor ligand, which binds a cognate non-estrogen receptor to modulate estrogen activation of an AP-1 site; contacting a cell with both a ligand to a cognate receptor and a compound having AP-1 mediated estrogenic activity wherein the ligand modulates estrogen activation at an AP-1 site.

The present claims are patentably distinct from the '291 claims because many of the claimed elements are not found in the suggested combination of references. Furthermore, there is no motivation to combine the prior art with a reasonable expectation of success, as discussed above. Therefore, the rejection for alleged double patenting should be withdrawn.

CONCLUSION

In view of the foregoing, Applicants believes all claims now pending in this application are in condition for allowance. The issuance of a formal Notice of Allowance at an early date is respectfully requested.

If the claims are deemed not to be in condition for allowance after consideration of this Response, a telephone interview with the Examiner is hereby requested. Please telephone the undersigned at (510) 769-3510 to schedule an interview.

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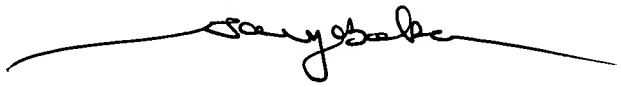
Respectfully submitted,



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Attachments:

1) A transmittal sheet; and,
2) A receipt indication postcard.

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